CLAIMS

1. A method of treatment or prophylaxis of a condition associated with elevated levels of non-amidated gastrin, comprising the step of administering to a mammal in need of such treatment an effective amount of a compound which has the ability to inhibit the binding of ferric ions to any one or more of glycine-extended $gastrin_{17}$ or progastrin or progastrin-derived peptides, but which does not inhibit the activity of amidated gastrin, thereby to inhibit the activity of non-amidated gastrins.

2. A method according to claim 1, in which the compound inhibits the binding of ferric ions to glutamate 7 of glycine-extended $gastrin_{17}$.

3. A method according to claim 2, in which the binding of ferric ions to glutamate 8 and glutamate 9 of glycine-extended $gastrin_{17}$ is also inhibited.

4. A method according to any one of claims 1 to 3, in which the compound is a metal ion, or a pharmaceutically-acceptable salt or complex thereof, which is able to occupy the ferric ion binding site of non-amidated gastrins, and thereby to block their biological activity.

5. A method according to claim 4, in which the metal ion is any metal ion capable of occupying the ferric ion binding site of non-amidated gastrins, with the provisos that

(i) when the condition is one caused by *Helicobacter pylori* infection, the metal ion is not bismuth, and

(ii) when the condition is cancer, the salt or complex is not $BiISrC_6H_5O_6$.

6. A method according to claim 5, in which the metal

ion is Bi^{3+} or Ga^{3+}.

7. A method according to any one of claims 1 to 3, in which the compound is an exchange-inert complex between a non-amidated gastrin and either Co (III) or Cr (III) ions.

8. A method according to any one of claims 1 to 3, in which the compound is a pharmaceutically-acceptable chelating agent with a high degree of specificity for ferric ions.

9. A method according to claim 8, in which the chelating agent is membrane-impermeable.

10. A method according to claim 9, in which the chelating agent is desferrioxamine (DFO), ethylene diamine tetracetic acid (EDTA) or diethylene triamine pentacetic acid (DTPA).

11. A method according to claim 8, in which the chelating agent is a membrane-permeable chelator.

12. A method according to claim 11, in which the chelating agent is clioquinol.

13. A method according to any one of claims 1 to 12, in which the compound does not have a significant inhibitory effect on Gamide-induced inositol phosphate production and/or on cellular proliferation in cells which express the CCK-2 receptor.

14. A method according to claim 6, in which the compound is one or more of colloidal bismuth subcitrate (CBS), bismuth subcitrate, bismuth citrate, bismuth salicylate, bismuth subsalicylate, bismuth subnitrate, bismuth subcarbonate, bismuth tartrate, bismuth subgallate,

tripotassium dicitrato bismuthate or bismuth aluminate.

15. A method according to claim 14, in which the compound is one or more of colloidal bismuth subcitrate (CBS), tripotassium dicitrato bismuthate, bismuth subcitrate, or bismuth subsalicylate.

16. A method according to claim 15, in which the compound is CBS or tripotassium dicitrato bismuthate.

17. A method according to claim 16, in which the compound is CBS.

18. A method according to any one of claims 1 to 17, in which the condition is selected from the group consisting of gastrin-producing tumours, colorectal carcinomas, gastrinomas, islet cell carcinomas, lung cancer, ovarian cancer, pituitary cancer and pancreatic cancer.

19. A method according to claim 18, in which the condition is colon cancer or pancreatic cancer.

20. A method according to claim 19, in which the condition is colon cancer and the mammal is at elevated risk thereof.

21. A method according to claim 20, in which the mammal is an individual with any one or more of familial adenomatous polyposis, with a family history of colon cancer, and/or with loss of imprinting of IGF-2.

22. A method according to any one of claims 1 to 17, in which the condition is selected from the group consisting of atrophic gastritis, G cell hyperplasia, pernicious anaemia, renal failure and ulcerative colitis.

23. A method according to any one of claims 1 to 17, in which the condition is selected from the group consisting of gastrointestinal ulcers, gastro-oesophageal reflux, gastric carcinoid, and Zollinger-Ellison syndrome, with the proviso that the metal ion is not bismuth.

24. A peptide which is a fragment of a non-amidated gastrin and which

(a) comprises at least glutamate residue 7 of the -$(Glu)_5$- sequence of non-amidated gastrin, and

(b) which is capable of binding one or more ferric ions, with the proviso that the peptide is not full length Ggly, full length glycine-extended gastrin or full length progastrin.

25. A peptide according to claim 24, consisting of amino acids 5 to 14 of the Ggly sequence.

26. A peptide according to claim 24, selected from the group consisting of $Ggly_{5-18}$, $Ggly_{1-11}$, LE_5AYG, LE_5AY, LE_5A, LE_5, E_5A, E_5, and E_5AY.

27. A peptide according to any one of claims 24 to 26, in which the carboxy terminus of the peptide is amidated.

28. A peptide according to any one of claims 24 to 26, in which the amino terminus of the peptide is acetylated.

29. A complex comprising

(a) a non-amidated gastrin, or a peptide fragment thereof according to any one of claims 24 to 28, and

(b) a trivalent metal ion.

30. A complex according to claim 29, in which the trivalent metal ion is Bi^{3+} or Ga^{3+}.

31. A complex according to claim 29 or claim 30, comprising a non-amidated gastrin and bismuth ions.

32. A pharmaceutical composition comprising
(a) a peptide according to any one of claims 24 to 28 or
(b) a complex according to any one of claims 29 to 31,
together with a pharmaceutically acceptable carrier, excipient or diluent.

33. A method of promoting intestinal function, comprising the step of administering
(a) a peptide according to any one of claims 24 to 27 and/or
(b) a complex according to claim 28 or claim 29
to a subject in need of such treatment.

33. A method according to claim 31, in which the subject is suffering from injury to the bowel, an inflammatory condition of the bowel, or short bowel syndrome, has undergone a partial or complete resection of the bowel, or is undergoing total parenteral nutrition.

34. A method of screening of candidate metal ion-binding compounds for ability to modulate the activity of non-amidated gastrins, comprising the steps of
a) assessing the ability of the compound to inhibit binding of ferric ions to a non-amidated gastrin and/or
b) assessing the ability of the compound to modulate proliferation and/or migration of cells of a gastric mucosal cell line in response to a non-amidated gastrin.

35. A method according to claim 34, in which the non-amidated gastrin is $Ggly_{17}$.

36. A method according to claim 34 or claim 35, in which the gastric mucosal cell line is IMGE-5.

37. A method according to any one of claims 34 to 36, in which the compound is additionally assessed for its ability to inhibit Gamide-induced inositol phosphate production, and/or cellular proliferation in cells which express the CCK-2 receptor.

38. Use of a compound which has the ability to inhibit the binding of ferric ions to glycine-extended $gastrin_{17}$ or to progastrin, but which does not inhibit the activity of amidated gastrin, in the manufacture of a medicament for the treatment or prophylaxis of a condition associated with elevated levels of non-amidated gastrin.

39. Use of

(a) a peptide fragment according to any one of claims 24 to 27 and/or

(b) a complex according to claim 28 or claim 29

in the manufacture of a medicament for promoting intestinal function.